express transcription factors including NFATc1, WT1 and Tbx18. To what extent these factors correlate with cell fate in EPDCs is unknown. **We hypothesize that the localized expression of NFATc1, WT1 and Tbx18 in PE cells and EPDCs influences EPDC cell fate.** Chick embryonic day 4 (E4) PE and E7 epicardium were explanted to compare NFATc1, WT1 and Tbx18 function in PE cells and EPDCs. NFATc1, WT1 and Tbx18 are expressed in subsets of cultured PE cells and EPDCs. Therefore, these cultures will be used to investigate cell type diversity in PE cells versus EPDCs. In a subset of cultured PE cells, NFATc1 colocalizes with the endothelial marker Flk1, which suggests that NFATc1 marks endothelial precursors *in vitro*. Tbx18 and WT1 are expressed in subsets of EPDCs, but the fate of these cells is unknown. Also, loss of Tbx18 function using Tbx18 specific siRNA significantly decreases EPDC proliferation, as detected by BrdU incorporation assay. These data suggest that NFATc1, WT1 and Tbx18 mark different cell lineages in PE and EPDC cultures, and that Tbx18 regulates EPDC proliferation. Our long-term goal is to define the molecular mechanisms that regulate PE and EPDC proliferation and cell lineage specification.

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**Program/Abstract # 308**
**Agtrl1b acts non-cell-autonomously for proper cell migration during myocardial progenitor development**
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During vertebrate embryogenesis the heart is the first organ to form and function. Fate-mapping studies in vertebrate embryos have demonstrated that prior to gastrulation myocardial cells originate from a fixed position in the embryo. During gastrulation these cells migrate to form bilateral stripes in the anterior lateral plate mesoderm (ALPM), where they first express *nkx2.5*, the earliest marker of myocardial progenitors. How cardiac cell fate is influenced during gastrulation remains unclear. A zebrafish mutant, *grinch*, in which there is a significant reduction (or complete absence) in the number of cardiomyocytes formed has previously been described by our lab. The *grinch* phenotype is due to a mutation in the gene encoding the G protein-coupled receptor Agtrl1b. Here we investigate the mechanism through which Agtrl1b regulates the formation of myocardial precursors. RNA *in situ* hybridization analyses of *grinch* mutants and morphants show that there is a decrease in specific domains of the ALPM. Lineage tracing studies demonstrate that this is likely due to aberrant cell migration during gastrulation. We find that in morphant embryo cells of the presumed heart field fail to reach the ALPM. Additionally, transplant studies reveal a non-cell-autonomous role for the function of Agtrl1b in myocardial progenitor development. Present studies are centered on imaging migrating progenitors in real time to examine specific defects in *grinch* mutants. Our work provides novel insight into the earliest mechanisms that influence cardiac progenitor development.

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**Program/Abstract # 309**
**FGF/Ets target genes in *Ciona intestinalis* heart cell specification**
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Activation of the transcription factor Ets1/2 through FGF signaling is known to specify heart precursor fate in *Ciona intestinalis*. In previous research, we identified candidate target genes of Ets1/2 through microarray analysis. Through in situ hybridization assays we have identified a subset of these candidate genes that are expressed specifically in the heart precursor cells immediately following their specification. To find the enhancers for the regulation of these presumed Ets target genes, we are employing bioinformatics to find conserved areas of DNA in the upstream non-coding DNA between *C. intestinalis* and *Ciona savignyi*. This analysis will be used to guide ongoing efforts to clone and test predicted enhancer regions using reporter constructs. In depth analysis of identified enhancers will be used to find transcription binding sites for Ets and identify co-transcription factors presumed to act in concert with Ets to drive heart precursor cell specification.

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**Program/Abstract # 310**
**FGF signaling regulates spindle dynamics in *Ciona* heart precursor cells**
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Asymmetric cell division is a fundamental mechanism in developmental biology by which a single fertilized cell can develop into a multicellular organism. Previous studies have shown that asymmetric divisions are typically caused by a shift in spindle orientation and position. In *Ciona intestinalis*, one such division is required to establish the heart precursor cells. Each of the four B7.5 lineage cells (founder cells) divides asymmetrically to produce a large tail muscle cell and a smaller heart precursor cell. Previous research has shown that this asymmetric division requires a non-polarized FGF signal from the adjacent mesenchyme, which results in uniform FGF receptor occupancy on the B7.5 cells. This causes a localized change in cytoskeletal dynamics resulting in asymmetric division of the B7.5 lineage. Embryos treated with a dominant negative form of the FGF receptor undergo symmetric founder cell division. Also, polarity gene manipulation (constitutively active Cdc42) and inhibition of MAPK signaling result in loss of founder cell asymmetry. To better understand how these molecules interact and their direct effect on division symmetry, we are using live fluorescent microscopy to investigate spindle dynamics within the founder cell. In addition, we are using molecular cloning techniques to identify target candidate molecules that might also be involved in this pathway. While the effect of spindle dynamics on asymmetry has been well studied, no previous study has linked FGF signaling to asymmetric division and the mechanism of cell division symmetry in vertebrate heart precursor cells has yet to be understood.

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**Program/Abstract # 311**
**How heart cells embrace their fate in the chordate *Ciona intestinalis***
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The establishment of polarity and subsequent asymmetric cell division is required for differentiation throughout development. In *Ciona intestinalis,* such a division occurs in the heart founder cells, with each of four founders giving rise to a small heart progenitor cell and a larger tail muscle cell. Although FGF signaling occurs prior to division, ERK is activated only in the smaller daughter and results in heart cell-specific behaviors such as migration and proliferation. The mechanism by which FGF signaling is propagated only in the heart lineage is not yet understood. Our data implicates polarity of the actin cytoskeleton in